fore," or "we were not told to do it in medical school." We just haven't thought of it often enough. But the advantages are worth the effort of writing an extra five-letter word, or the print on the prescription blank, and the scratch where it does not apply.

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Single Injury and Cancer

IN AN ARTICLE entitled, "Can a Single Injury Cause Cancer?," by Dr. Arden R. Hedge in the January 1959 issue of CALIFORNIA MEDICINE, it is my belief that the problem is discussed superficially and that numerous recent concepts relating to the pathogenesis of cancer are ignored. I note that except for a reference to a review article in *Cancer Research* concerned with the basic aspects of cell division the most recent reference in the bibliography is that to an article published by Dr. F. W. Stewart in 1944. During the intervening years, experimental and clinical observations suggest that the concept of trauma in cancer certainly is in need of review. The conclusions of Stewart[1] that "Attempts to rely on single trauma to explain cancer depend on the exercise of primitive forms of reasoning," and Downing[2] "—I have never been thoroughly satisfied that a single trauma ever caused cancer—" are in need of re-examination in light of recent investigations on the role of skin sensitization and cocarcinogenesis in the etiology of skin cancer. Special emphasis should be directed toward the part played by wound healing in its action as a promoting agent or cocarcinogen.

The concept of "sensitization" or "preparation" of skin by means of suboptimal exposure to carcinogenic hydrocarbons has been experimentally established by Berenblum,[3] Berenblum and Shubik,[4] Rous and Kidd,[5] and Friedewald and Rous.[6] The suboptimal exposure serves as an initiating phase by converting some of the cells in the skin of experimental animals to a preneoplastic condition. Following this stage, which Berenblum refers to as precarcinogenesis and which Rous refers to as the stage of initiation, nonspecific agents such as wound healing, freezing with carbon dioxide snow, croton oil, and mechanical irritation are capable of converting the skin to true neoplasm. Friedewald and Rous, in their experiments on rabbits, showed that wound healing may act as a promoting agent. Shubik in attempting to confirm this observation modified their technique and succeeded in producing skin papillomas at the site of the induced trauma. No malignant changes were observed at the time the animals were sacrificed. He did conclude, however, that wound healing was undoubtedly effective as a promoting agent.

The carcinogenicity of certain petroleum oils that are obtained from the fluid catalytic cracking process has been demonstrated by Holt and his co-workers[7] in experiments on mice, rabbits, and monkeys. They further concluded that, "Employees exposed to contact with these oils are believed to be exposed to an occupational cancer hazard."

A case reporting the "Possible role of trauma as a cocarcinogen" in an oil worker by Kotin and Kahler[8] was recently published. Shimkin and his associates[9] reported the appearance of a carcinoma following exposure to a refrigeration ammonia-oil mixture. They concluded, "In our opinion a causal connection can be reasonably postulated between the trauma and the exteriorization of a latent neoplasm as an example of a cocarcinogenic effect." Smith[10] in a discussion of pulmonary cancer stressed that not only carcinogenic materials have to be considered but also cocarcinogenic agents which may be related or unrelated to the evoking agent.

An ever-expanding list of actual or potential carcinogenic agents is being introduced into the occupational environment as the result of newer industrial processes and the increased use of petroleum and its by-products. While exposure is admittedly kept at a minimum by industrial health control measures, suboptimal exposures to carcinogenic agents do occur with attendant danger of establishing the stage of initiation. In this light, the routine dismissal of trauma as a noncontributing factor to carcinogenesis should be replaced by the taking of a detailed occupational and environmental history of the patient to see if the process of cocarcinogenesis may have been a factor.

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